Leaming (J. R.)

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DISTURBED ACTION AND FUNCTIONAL MURMURS

OF THE

H E A R T.

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J. R. LEAMING, M. D.,

PHYSICIAN TO ST. LUKE'S HOSPITAL, NEW YORK.

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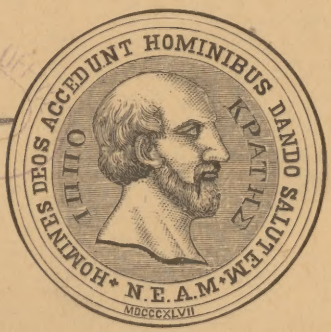

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SIGNIFICANCE OF DISTURBED ACTION AND FUNCTIONAL MURMURS OF THE HEART.

By J. R. LEAMING, M. D.,
PHYSICIAN TO ST. LUKE'S HOSPITAL, NEW YORK.

Read March 18, 1875.

In April, 1868, I had the honor of reading a paper on "Cardiac Murmurs" before the New York County Medical Society, in which my endeavor was to substantiate the true diagnostic sign of mitral regurgitation; and also the significance of intra-ventricular or mitral non-regurgitant murmurs, as were held by my friend the late Dr. Cammann. He had demonstrated, by pathological investigations, that the signs of mitral regurgitation as generally taught—murmurs at the apex beat, blowing, sawing, rasping, etc.—were unreliable, but that the true and invariable sign is a murmur of an entirely different character—a soft murmur, a friction-murmur, such as would naturally be formed by forcing fluids through an aperture, and which is heard behind, between the seventh and eighth vertebræ of the left side, close to their spines; and that, with this sign alone, mitral regurgitation is with certainty diagnosticated. The mechanism of the first sound is evidently the key to a correct diagnosis of a large majority of murmurs, both functional and organic. The theories of the cause of the first sound, according to Bellingham, "may, for convenience' sake, be considered, as the cause is supposed to be extrinsic or intrinsic to the heart. Thus under the first, it has been attributed to the impulse of the apex against the parietes of the chest; under the second head, it has been attributed to muscular contraction — in other words, to the successive shortening of the muscular

fibres of the parietes of the ventricles. This is the oldest theory; it was adopted by Harvey, Haller, Senac, Bichat, and Corvisart. 2. To the sudden tension of the auriculo-ventricular valves. 3. To the friction of the blood against the parietes of the interior of the ventricles, or of the orifices of the large arteries. 4. To the collision of the opposite internal surfaces of the ventricles at the conclusion of the systole. 5. To the sudden elevation of the sigmoid and semilunar valves, caused by the wave of blood transmitted by the ventricles. 6. To the concussion of the blood transmitted by the systole of the left ventricle, with that contained in the aorta; and, lastly, to two or more of the foregoing causes combined."

I chose to consider as worthy of attention only three of the theories in vogue: 1. That of friction of the blood in its motion, within the ventricle, and its passage into the aorta. 2. That of the muscular contraction of the heart itself producing sound-vibrations, as shown by Dr. Wallaston, in 1810; and, 3. That of the vibrations of the mitral valve, caused by its closure and tension, and the forcing and rushing blood; and lastly, that some, recognizing the possibility of each of these three causes mentioned producing sound, have believed that, as the first sound is evidently composite, it is the result of all three. This was the theory held by Dr. Cammann.

As none of these theories seemed to me to agree with all the conditions, and especially with acoustical conditions, I was impressed with the truth that they did not give satisfactory evidence of the cause of the first sound, and that we must direct attention to the heart itself for new proof on this vexed question. We find a peculiar musical-instrument arrangement within the heart, of a drum-like expansion of fibrinous tissue, to which are attached fine, tendinous cords, joining each part of the valve to the wall of the heart, through the intervention of bundles of muscular fibres—columnæ carneæ, or musculi papillares. It seems incredible that such admirable conditions for producing sound-vibra-

tions could have so long been overlooked by the many able observers, as the most probable cause of the first sound.

That the first sound is caused by vibrations of the chordæ tendinæ, connected with the mitral valve in the left heart, and with the tricuspid in the right, set in motion by the swift current of forced blood, is a reasonable postulate. If this doctrine can be proved by pathological evidence of undoubted character, it simplifies our investigation. If plastic lymph be exuded upon the surface of the valve, or upon its edges, gluing them together, and if at the same time the chordæ tendineæ are shortened and thickened by exuded plastic lymph, or glued down upon the valve so as to prevent vibration, then, if the first sound is altered, and all murmurs are abolished, it must be admitted that the proof is sufficient. The following cases are offered as supplying such convincing evidence:

Case I. *July* 6, 1859.—John Martin: is a native of England; educated at Eton; forty-two years old; during the last ten years has been dissipated, and has had syphilis; had rheumatism eight years ago, which kept him in bed two weeks; and has since had frequent rheumatic pains; with these exceptions, has been well until about two years ago, when his appetite failed, and he vomited mornings after taking beer; and his weight declined from one hundred and ninety to one hundred and forty-four pounds. Two days ago, while at his business, there was momentary loss of consciousness without falling, and similar attacks occurred frequently until last night, when they prevented sleep.

Examination.—The pulse grew gradually weaker, until it could no more be felt, and at the same time the respiration would be suspended. The interval was so long, that I looked in his face to see if he were not dead; when, with a full inspiration, and a strong throb of the pulse, both would commence again and continue about fifteen pulse-beats, then cease, and begin again as before. In addition to this were the attacks of "petite mal"— his face would flush slightly, and his eyes stare as if he saw a strange object—this would

scarcely interrupt his conversation, when he would go on again as if nothing had happened. These epileptiform seizures came during the intermissions of the pulse and breathing, as well as at other times.

Auscultation of the chest discovered no fault in respiratory murmurs. There was a slight systolic cardiac murmur, aortic-obstructive. After an intermission of the heart-beat, which agreed in length with the intermission of the pulse, it would begin again with a forcible impulse, which gradually decreased in strength until it ceased to be felt or heard, after which one contraction of the heart could be heard, but without first sound or impulse. The sound of this contraction was peculiar; it was as if no blood was forced into the aorta by ventricular contraction. By careful counting, repeated a number of times, the exact time of the heart's rest was found to be sixteen seconds. The heart seemed to beat in a wild and peculiar manner, as if outside of the pericardium, and the point of impulse varied an inch or an inch and a half.

The next day Dr. T. M. Halstead was called as counsel, the conditions remained unchanged.

8th.—Was called at 6 A. M to see the patient, who was supposed to be dying. I was informed that an intermission of extraordinary length had occurred. Respiration and pulsation had ceased, the hands fell by his side, his chin dropped, his head inclined to one side, and his face became livid. His sister, who sat by him, believing him to be dying, called his wife; her outcries awakened him, and after a short time he recovered, and was as he had been before. When I arrived his pulse was 25 in the minute, as it had been from the first, and his state remained unchanged in both signs and symptoms.

Friday, 10*th*, 7 P. M.—Dr. Alonzo Clark was added to the consultation. Dr. Clark found the time of intermission of the pulse to be thirteen seconds; the seizures are a little more violent, and he is nervous. Physical signs the same as before.

11*th*.—Patient has slept during the night. The epilepti-

form seizures ceased at midnight, and the pulse has become regular without intermissions—52 in a minute. After this the patient steadily improved, and one month afterward he walked to Dr. Cammann's office in Fourth Avenue. Dr. Cammann diagnosticated systolic obstructive murmur, with hypertrophy of the heart, but believed the irregular action and peculiar symptoms were owing to functional derangement from indigestion. He became well enough to attend to business until October, 1861, when he was again taken ill. There were anasarca, dyspnœa, and laboring heart and obscure physical signs. He gradually failed, and died on November 26, 1861.

Post mortem on 27th, assisted by Dr. Loomis. Complete adhesion of the pericardium to the heart. There was no free space, but in some parts the adhesions were stronger and apparently older than in others. The heart was largely hypertrophied, but was not weighed. The curtains of the aortic valve were thickened and shortened to incompetency, not holding water. The edges of the mitral valve were glued together, extending into the ventricle like a funnel: complete stenosis. The opening very small, the valve and chordæ were thickened and covered with plastic lymph, white and glistening.

Case II. (*Substance of Remarks made by* James R. Leaming, M. D., *before the Pathological Society on the Presentation of a Specimen for a Candidate for Admission.*)—Mrs. B——, twenty-three years of age, native of New York, widow, called Dr. S——, in April, 1869, for advice as to cardiac trouble and swelled feet. The doctor found, on examination, a systolic murmur over the base of the heart, more distinct over the aortic valves, gradually disappearing to the right in the course of the aorta; there was also a diastolic murmur.

Diagnosis.—Aortic obstruction and aortic regurgitation, with hypertrophy of left ventricle. There were also casts in the urine and albumen. She became dropsical, her condition gradually grew worse, and she died in September last.

I saw the case with Dr. S——, in May, and found no dif-

ferent conditions than those already discovered. *There was no mitral murmur of any kind.* The specimens here presented show Bright's small kidney of advanced disease. The heart is hypertrophied mostly in the left ventricle; the aortic valve is thickened at the base of the curtains; shortened to incompetency—so far, agreeing with the diagnosis. But the mitral valve presents the most notable feature. There was no sign of disease of this valve during life, and yet it is damaged in a very peculiar manner. It is thickened by lymph-deposit; its color white, opaque; the edges of the curtain are adherent, and the orifice is narrowed down till it will barely admit the top of the index-finger; and the whole valve extends down into the cavity of the ventricle like a funnel. The chordæ tendineæ were shortened and thickened by lymph-deposits, and the musculi papillares were thickened and lengthened. But every thing was symmetrical, viz., the funnel-like condition of the valve, the hypertrophy of the cardiac walls, of the musculi papillares, and of the columnæ carneæ. With perfect conditions for producing a *mitral direct murmur*, it was absent.

Case III. (*Copied from Reports of the Pathological Society, published in the Medical Record in* 1871.)—Dr. Loomis presented a heart, with the following history, from Dr. Milliken, house-physician of Bellevue: "Henry Clemens, admitted April 11, 1871, aged thirty-two; single; cabinet-maker by occupation; nativity, Switzerland. Patient gives hereditary history of pulmonary phthisis. Had an attack of articular rheumatism when seventeen years of age, from which he made a good recovery. States that neither at that time, nor since, has he experienced any precordial pain, but has noticed that after indulging in tobacco (for he has been an inveterate smoker) he would suffer from palpitation of the heart. He had had a cough, dating some time back, with some expectoration of a pearly white material, which he says he coughs up at night, at which time his cough distresses him most. About two weeks ago, for the first time, he noticed that the sputa were streaked with blood. His cough remained about

the same in character, until one week ago, when he experienced a severe paroxysm of coughing, which was instantly followed by hæmoptysis, which continued for two or three days. Since the occurrence of hæmoptysis, he has had night-sweats, loss of appetite, depreciation of strength, and experienced a feeling of general *malaise*, and inaptitude for any kind of work; he complains also of insomnia and restlessness. His pulse is about 80, regular, but quite feeble; respiration somewhat hurried and easily performed. Heart: action regular, but *quite feeble;* apex-beat on a level with nipple in fifth interspace. Heart-sounds *feeble;* after repeated examinations, no *murmurs could be detected.*"

The record proceeds to say that, while the patient was at dinner, he became suddenly unconscious and fell from his chair, and symptoms of paralysis continued until the 18th, when he died. *Post mortem* showed embolism of middle cerebral artery of left side, with softening of brain-tissue. "Heart, fourteen ounces. Both right and left cavities contain large clot of blood; substance of heart relaxed; stenosis of mitral orifice only admits little finger; some shortening of chordæ tendineæ. The stenosis is due particularly to the thickening, shortening, and adhesion, of the chordæ tendineæ of the valve. The anterior portion of valve forms a bony mass, occluding that portion of the orifice. On the auricular aspect, the surface of the valve is ulcerated, the bony matter laid bare, and soft, reddish vegetations on the free border of the valve and upon the ulcerated surface. Pulmonary and tricuspid valves normal; little thickening at base of aorta."

Dr. Loomis remarked, "The case is of special interest, because with this marked stenosis no murmurs existed;" and Dr. Flint remarked that "the absence of murmurs might be accounted for—1. On account of rigidity of the valve not allowing a vibration; and, 2. The smoothness of the ventricular surface of the valve."

The first case is full of instruction in its facts as regards functional disturbances of the heart and proof as to the mechanism of the first sound. The long period of rest, six-

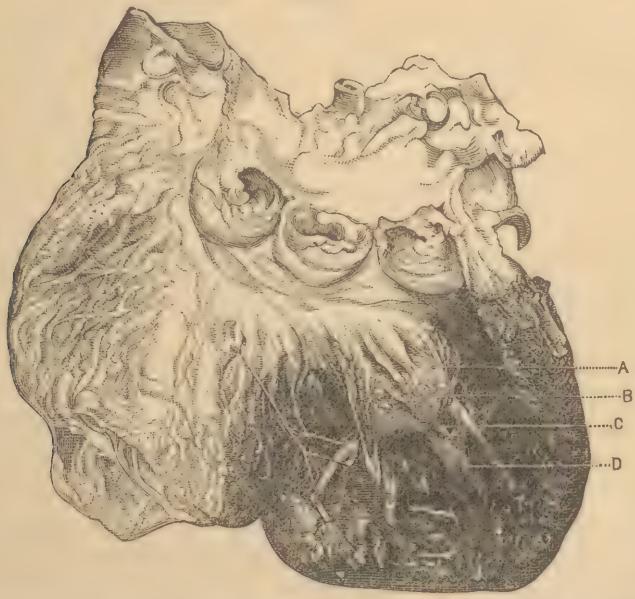
teen seconds, is worthy of our earnest attention. Observers who have watched the action of the heart in ectopia in an infant, as Cruveilhier, Bryan, and others, as well as when the heart has been exposed in experiments upon animals, tell us that the contractions of the auricles continue regularly, although the ventricles may be in a state of rest. And in this case no doubt they did so, notwithstanding that there was no first sound, no impulse-beat, and consequently no contraction of the ventricles. The importance of this fact cannot be over-estimated, because it invalidates much of the theory in vogue in regard to the causation of murmurs. It proves that the auricular systole may take place regularly, even when the auriculo-ventricular opening is very much contracted in stenosis of the mitral valve, without producing sound. Carefully listening under favorable circumstances after the last impulse-beat and first sound, one contraction, presumably that of the ventricle, could be heard, without any vocal element of first sound, and was then followed by the long interval of silence, in which no contraction or sound of any kind could be heard.

The second case is a demonstration of the cause and mechanism of the first sound. *There was no mitral murmur.* With stenosis of the mitral valve, if the chordæ tendineæ had not been rendered incapable of sound-vibrations, by being plastered over with fibrinous deposit, there would have been a murmur, such as is usually heard in stenosis where the chordæ are free and uncovered. The first sound, and all murmurs connected with it, disappearing when the mitral valve and chordæ tendineæ are rendered incapable of sound-vibrations, is as convincing proof of their cause, as is the experiment of hooking up a curtain of the aortic valve proof as to the cause of the second sound.

The second and third cases are confirmatory proof, by different observers, that the cause and mechanism of the first sound and the murmurs connected with it, depend upon the state and condition of the mitral valve and its chordæ tendineæ. In the second case there was no physical sign of dis-

ease of this valve during life, and yet it was found after death to be damaged in a very peculiar manner—thickened by lymph-deposits, opaque, its color white, the edges of the curtains adherent, the orifice narrowed down, barely admitting the tip of the index-finger, and the whole valve extending down into the cavity of the ventricle fixed and like a funnel. The chordæ tendineæ were shortened and thickened, some of them glued to the valve, and the musculi papillares thickened and lengthened, as the specimen which I now present to you

Fig. 1.



A, Stenosis of the mitral valve; *B*, Chordæ tendineæ glued to the valve; *C*, Chordæ tendineæ shortened and thickened; *D*, Hypertrophied and lengthened musculæ papillaria.

demonstrates. This case, during several months, was under the observation of the late Dr. Sprague, a careful and competent auscultator.

The third case, which is reported in the Transactions of the New York Pathological Society, is also confirmatory proof: "In the morbid specimen there was stenosis of mitral

orifice—only admits little finger—some shortening of chordæ tendineæ. The stenosis is due particularly to the thickening, shortening, and adhesion of the chordæ tendineæ of the valve." During life, "heart-sounds feeble; after repeated examinations no murmurs could be detected. Could the proof be more conclusive?

The following experiments by Dr. Halford, quoted in the *British and Foreign Medico-Chirurgical Review*, April, 1860, is singular proof of the physiological cause of the first sound: "My proceedings were as follows: large dogs were obtained, and as in my preceding experiments (the animals being under the influence of chloroform), the heart was exposed and the circulation kept up by artificial respiration. A stethoscope being applied to the organ, the sounds were distinctly heard. The superior and inferior venæ cavæ were now compressed with bull-dog forceps, and the pulmonary veins by the finger and thumb; the heart continuing its action, a stethoscope was again applied, and neither first nor second sound was heard. After a short space of time, the veins were allowed to pour their contents into both sides of the heart, and both sounds were instantly reproduced. The veins being again compressed, all sound was extinguished, notwithstanding that the heart contracted vigorously. Blood was let in, and both sounds were restored. I have thus frequently interrogated the same heart for upward of an hour, and always with the like result."

The reviewer remarks: "There is an interesting circumstance which took place at one of Dr. Halford's experiments, which appears to us of great importance. It shows that when only a small quantity of blood finds its way into the ventricles, the first sound is still produced. The cavæ and pulmonary veins having been compressed, Mr. Lane, at whose request the experiment was performed, listened to the heart during its contraction, and said he heard the first sound indistinctly, not so clearly as before the compression. On examination it was found that the vena azygos entered the right auricle by an independent opening, and was not se-

cured: the vessel was compressed with the others, the heart contracted, no sound was heard."

This experiment proves that the contractions of muscle of the heart give out no sound which may be an element of the first sound; for without blood moving through the heart it was silent. The remaining elements, friction of the blood against the heart-wall and through the aortic orifice, and vibrations of the chordæ tendineæ and mitral valve, must give answer to the question. When there was no blood forced there was no sound; and we have just shown, by pathological specimen, that when the chordæ tendineæ were rendered incapable of vibration, there was also neither sound nor murmurs. Consequently, the first sound and murmurs must be the result of chord and valve vibrations set in motion by the rushing blood. The blood is the bow applied to the strings to give vibrating sounds; and murmurs are sounds of individual chord-vibrations not in unison.

One of the points I endeavored to establish in 1868 was, that the presystolic murmur, called also the auricular-systolic and the mitral direct, is one of the intra-ventricular murmurs, caused by vibrations of chordæ tendineæ subjected to irregular tension, and not by blood being forced through the contracted opening of the mitral valve in stenosis. Although frequently connected with that pathological condition, it is yet oftener an accompaniment of change of the mitral valve without stenosis. My argument was, as Dr. Cammann contended, that the auricle was too feeble a power to force blood through the contracted opening of the diseased valve, so as to cause sound which may be heard through the chest-wall, even if empty, much less so when the ventricle is filled with blood; and, lastly, I maintained that the murmur does not agree in length with the time of contraction of the auricle. According to the best authorities, the contraction of the auricle is instantaneous, while the murmur is of considerable length.[1] If contraction of the auricle could cause the mur-

[1] Harvey, Lower, Bellingham.

mur, the two ought to agree in time. According to Bellingham, "the systole of the auricle is a quick, short, sudden motion." Lower says, "Its rapidity equals the explosion of gunpowder, and immediately precedes the ventricular systole, the one motion appearing to be propagated by the other."

Marey assigned to it two-tenths of the time of the heart-beat, which is probably ten times longer than the reality, and much less than the time of the so-called presystolic murmur. This murmur, too, has none of the qualities of sound which should be produced by blood forced through a narrowed opening in the valve. But all argument becomes unnecessary in presence of the foregoing pathological facts and clinical history. Dr. Frank Donaldson, Professor of Physiology and Hygiene, and Clinical Professor of Diseases of the Chest and Throat, University of Maryland, in a paper read before the Medical and Chirurgical Faculty of Maryland, annual session, April, 1874, on "Significance of the Presystolic Murmur," relates the following cases, with remarks:

"Some years ago (in 1867), a case came under my observation, which made me question the explanation which I had adopted on the authority of Barth, Roger, Walshe, and Flint, of the sound which was described first by Fauvel, in 1843, and then by Grisolle, as the presystolic murmur, afterward by Dr. Gairdner, of Edinburgh, as the auricular-systolic murmur, and by Dr. Austin Flint as the mitral direct murmur.

"These authorities claimed that this sound was heard just *preceding* the ventricular contraction, and was caused by the systole of the auricle forcing the blood into the ventricle, through a diseased and contracted auriculo-ventricular orifice.

"The case was of a man sixty-four years of age, of grossly intemperate habits, who came to the Baltimore Infirmary with symptoms of advanced heart-disease—great dyspnœa, a small, contracted pulse, heart much hypertrophied, with a murmur of a rasping character, heard loudest between the

second and third ribs at the base, not extending up the carotids, but down toward the base, and completely obliterating the second sound of the heart. The murmur was audible after the apex-beat and the systole of the ventricle, and was followed by the pause of the heart. The first sound of the heart was normal. The diagnosis seemed clear and unmistakable, and was recorded as insufficiency of the aortic orifice, by means of which the arterial blood was forced back into the left ventricle.

"The *post mortem* showed atheromatous degeneration in the aorta above the semi-lunar valves extending to the sacs of Valsalva, and causing adhesion of one of the semi-lunar pouches of the aortic orifice to the wall, so bending it down that that portion of the orifice was unprotected. The second sound could not be produced, and the insufficiency of the valve was evident.

"Thus far the diagnosis was correct, but on examining the mitral orifice we found, to our surprise, that it was reduced by thickening at its base to about the size of one-quarter of an inch in diameter. Yet, during life, there was no abnormal sound preceding or during the ventricular systole. With such a contraction of the left auriculo-ventricular orifice, ought we not to have had a decided presystolic murmur? The whole heart, auricle and ventricle, was enlarged and increased in force, and yet there was no murmur produced from the passage of the blood through an orifice so reduced in size! I could not help questioning the received opinion as to the significance of the so-called mitral murmur. As it is a physical sound, heard at a particular period of the heart's action, the physical cause which was said to produce it being present, it ought to have been heard, but it was not.

"Hope, as far back as 1842, reports a case where the mitral orifice was so contracted that it would only admit the little finger, yet there was no murmur during life, preceding the first sound. In his report he adds: 'I have frequently known a contraction of the mitral orifice to the size of only two or three lines, to occasion little or no murmur.' Dr.

Stokes, in his work on 'Diseases of Heart and Aorta,' relates two cases of extreme contraction of the mitral orifice found after death, but where, during life, there had been no murmur audible even to his practised ear.

"Dr. Waters. His first case was where he heard a loud systolic as well as a presystolic murmur. At the autopsy there were found insufficiency and slight contraction of the mitral orifice. In his second case there was no presystolic murmur whatever, although the autopsy showed a constricted mitral orifice only admitting the tip of the index-finger. Next follow the details of four cases of extreme contraction of the mitral orifice, where, during life, there was no presystolic murmur audible. He candidly adds: 'I have given you instances sufficient to prove that great constriction of the mitral orifice may exist without there being any murmur produced by the passage of the blood from the auricle into the ventricle, and therefore that you must not look for a mitral-diastolic or presystolic as a constant sign of obstructive mitral disease. My belief is, that this murmur is far more frequently absent than present, even when there is great obstruction at the mitral orifice.' Dr. Waters accounts for the presence or absence of this murmur, as depending on the greater or less vigor with which the auricle contracts."

Dr. Donaldson sums up his relation of cases and remarks: "Thus we have eleven cases of the lesion without the murmur, and three cases of murmur without the lesion" (quoting the latter from Dr. Flint).

The diagnostic sign of mitral regurgitation, which has been and is still taught, is a harsh, blowing, sawing, or filing murmur, heard during the systole at the apex-beat. Upon the accepted authority of this murmur, which is so often met with, the great frequency of mitral insufficiency has come to be considered as incontrovertibly established.

The cases we have already related are proof that these murmurs are not heard when the chordæ tendineæ and valve are rendered unfit for sound-vibrations. J. S. Bristow, M. D., London, F. R. C. P., Physician to St. Thomas's Hospital, in

an article on "Mitral Regurgitation, arising independently of Organic Disease of the Mitral Valve," in the July number of the *British and Foreign Medico-Chirurgical Review* of 1861, gives six cases, with introductory remarks. With your permission I will read some of his arguments and quote points in the cases, for the purpose of showing that instead of proving that regurgitation may take place through the mitral valve without disease, as he imagines, they in reality disprove the theory in vogue, and confirm the doctrine of chordæ tendineæ vibrations as cause of the first sound.

Dr. Bristow remarks: "It may almost be regarded as an axiom in medicine, that the presence of a systolic apex-murmur is positive proof of regurgitation through the mitral orifice. I have not hesitated to adopt it in reference to the cases already detailed." The following are quotations from his cases:

Case I.—There was a distinct systolic murmur audible at the apex of the heart.

Post mortem.—The aortic and mitral valves were perfectly natural.

Case II.—There was an increased area of dullness in the cardiac region, and a systolic bruit loudest at the apex of the heart.

Post mortem.—The muscular tissue and the valves appeared perfectly healthy.

Case III.—The impulse was diffused and heaving, but not very strong. A systolic murmur was detected at the apex of the heart.

Post mortem.—All the valves were healthy-looking.

Case IV.—First sound at the apex was flapping and prolonged.

Post mortem.—The valves were perfectly healthy in texture.

Case V.—The cardiac dullness was enlarged, and a systolic murmur was audible with the heart's action, most distinct at a point an inch below, and internal to the left nipple.

Post mortem.—All the valves appeared perfectly healthy.

Case VI.—There was a distinct but not very loud systolic murmur, loudest in the usual situation of the apex of the heart.

Post mortem.—The aortic and mitral valves were perfectly healthy-looking, and doubtless quite competent.

A tabular arrangement like the following, in classifying murmurs acoustically, may be useful:

Valvular (all organic).	Aortic obstructive systolic.
	Aortic regurgitant diastolic.
	Mitral regurgitant systolic.
Intra-ventricular (more or less functional).	Organic functional.
	Inorganic functional.

These two great divisions are made in accordance with their acoustic differences. The sound in valvular murmurs is a friction-murmur, that of blood forced through an aperture. The intra-ventricular murmurs are mostly and distinctly chord-vibrations. The contraction of the muscular walls of the heart and its fleshy columns, the friction of rushing blood among the chordæ tendineæ and against the tense mitral valve, being the occasion of sound-vibrations, but is not the mechanism of the sound itself. As great difference exists between these murmurs as between that of a whisper and that of the voice. The obstructive systolic aortic may be modified by irregular calcifications in the aortic valves, extending into the column of forced rushing blood. In this way a harsher character may be given to the murmur, or it may even become musical. Vegetations also attached to the orifice or valve may be thrown into vibrations in the column of blood, and produce a musical murmur, but these are rare, mere possibilities. When musical murmurs occur they are almost always, if not always, vibrations of the chordæ tendineæ, some of which are under extraordinary tension.

These sounds or murmurs may be illustrated by a stringed musical instrument. Every degree in quality of murmur or sound from the softest blowing, up to the harshest, sawing, rasping, filing, or when the vibrations become sufficiently rapid and regular, into musical sounds. The use of the term

"bellows sound" by Laennec was unfortunate as applied to the murmurs of the heart, and much of the misunderstanding of murmurs and their mechanism is due to it. It is true that it describes the friction-murmur of blood forced through an aperture as in aortic regurgitation. It is like the sound of the air forced through the bellows; but the bellows-sound is not so like the friction-murmur, of blood forced through an aperture, as is fluid forced through an elastic syringe, in which some obstruction is created by pressure upon the tube. But, to imitate the murmur exactly, a fissure should be made in the bulb of the syringe, and then compressing it with force, the fluid escaping will give the exact sound. The only friction-sounds in cardiac murmurs proper are where the blood is forced through apertures or past obstructions; it is heard at the aortic orifice when there is obstruction, as by lymph-deposits upon the valve. It is at first uncomplicated, the simple gushing sound. But in time the obstruction causes hypertrophy of the left ventricle, which, having taken place, irregular tension of the chordæ tendineæ is the result, and vibrations out of unison with the first sound are carried with the current of blood, and both occurring in the systole, are mixed together and form what is called the blowing murmur.

It is now a sound of mixed elements, friction of blood against a solid, and vibration of strings under irregular tension. In order to have an intelligent understanding of these murmurs, we must analyze them and separate the sources of sound. We are assisted in this by localizing the sources. The blowing, sawing, filing, rasping sounds have their origin and cause within the ventricle; they are intra-ventricular. Dr. Cammann called them mitral-non-regurgitant. They are heard over the base of the heart, but always with greatest intensity at the apex-beat.

Friction-sounds are heard best over the orifices or in the direction of the vibrating column of blood. The aortic systolic obstructive murmur is heard over the aortic valves, and in the course of the column of blood. The regurgitant aortic diastolic murmur is heard over the aortic orifice, and to

the left and toward the apex-beat. The mitral aortic-regurgitant is heard behind on the left side near the spine. In this direction the blood is forced in regurgitation through the mitral valve; impinging first against the auricular wall, lying against the œsophagus, and aorta, and intervertebral substance, it is conducted directly into the ear, giving the sensation of being shot into it.

It may be heard a short distance from this point conveyed through the chest-wall. It may be heard in front, at the apex-beat, by conduction through the substance of the heart, when there are no intra-ventricular murmurs to destroy it or take its place. The discovery of this absolute sign of mitral regurgitation belongs to Dr. Cammann, and his last professional thought was given to its consideration. It is one of the most certain of cardiac signs. This characteristic murmur, heard in the situation he has pointed out, is an unfailing sign of mitral regurgitation. It had been my opinion that this characteristic murmur was never heard in front at the apex-beat—as it certainly is not when the valve is diseased, and the loud intra-ventricular murmur drowns and supplants it.

But the following case shows that it may be heard both behind and before in congenital mitral insufficiency, without hypertrophy of the heart and without lymph-deposits upon the valve.

Case VII. (*December* 12, 1870).—W. S. R., New York, aged twenty-two; mason, living in Yorkville; is a fireman temporarily, and was a member of the old department. Has never been sick, except with chills and fever. Sent for examination by Dr. Charles McMillan, surgeon of the department. There is a systolic murmur at the apex-beat accompanying the first sound; it is a soft, gushing murmur, and can be heard in the chest-wall more to the left than to the right side. It is heard also with directness and greater intensity between the seventh and eighth vertebræ, left side behind, near the spine. The murmur is shot into the ear when placed over this point. It can be heard some distance to the left, conveyed

in the chest-wall. It can also be heard over some portions of the right lung posteriorly, at the inner angle of the scapula; also at the lower angle, being a faintly-conveyed sound.

One year after, examined him again. Signs unchanged. This murmur has the same quality in front as behind. It has none of the vocal element of apex-beat murmurs, usually described as diagnostic of mitral regurgitant murmurs. Yet I have no doubt that this murmur is caused by mitral insufficiency, which is congenital, without hypertrophy of the heart, and without disease of the mitral valve.

A great majority of cardiac murmurs, even of those accompanying organic disease of the heart, are in a manner functional. That is, the murmurs are not organic in the same sense that the valvular murmurs are; which are organic murmurs because the structural change in the valve is part of the mechanism of the murmur. Intra-ventricular murmurs, even when the result of structural change in the heart, may be considered functional, inasmuch as that they have their mechanism in vibrations of the chordæ tendineæ, which are themselves unchanged by any diseased action, but simply vibrate, giving out sound of high or low pitch, soft or harsh, feeble or loud, according to the degree of tension of the individual strings, and the force of the heart's contraction. The cause of irregular contraction of the heart-muscles may be from disturbed nerve power, as well as from organic change.

Functional murmurs proper may occur in the healthy heart, are transient, passing away with the subsidence of the cause, which may be anæmia, hyperæmia, sympathy with brain-disease, stomach, liver, or it may be from disorder of the nervous system, the influence of tobacco, coffee, tea, or any narcotic or stimulant having influence upon the organic life of the body, of which the heart is the centre and citadel.

Functional murmurs proper do not signify danger of sudden death, but nothing more alarms patients than disturbed action of the heart. When the heart seems to stop, and then to turn over and thump against the chest-wall, the

sensation is not a pleasant one, even to a medical philosopher. It is no wonder that it creates intense alarm in the lay patient, especially if accompanied by prolonged palpitation or faintings.

These conditions may be the forerunner of softening, or fatty degeneration, but they signify always that there is overdistention of the portal system, intermission of the heart-beat and pulse, may be present for years, and be merely the result of functional disturbance from chronic indigestion.

Intermissions of the pulse have been laid down in books as signs of heart-disease. Life-insurance companies, in printed forms, make it the duty of examiners to reject as unsafe those who have intermittent pulse. It is possible that this rule militates against the interest of the companies, and it certainly is a source of great alarm to the rejected applicant.

The sign, of itself, is no proof of heart-disease, but is proof of indigestion. It is true, cardiac disease is frequently a cause of indigestion, and thus, secondarily, the cause of irregular pulse. But a confirmed dyspeptic is usually a safe life, for he is not likely to commit indiscretions in diet, as he is continually warned to desist by functional disturbances. Proper medication will generally relieve intermittent pulse, even in advanced cases of cardiac disease.

A sedative dose of calomel will frequently set it right at once, and the intermissions will disappear.

The late Dr. Samuel Henry Dickson stated that, during the first hours of sleep, children have intermittent pulse, which will disappear when they are awakened. This is true, especially with those children who are allowed over-stimulating food, but, as the night passes on, and the food becomes digested, the intermissions cease. In the adult, the occasion of a wine-dinner, with tobacco, is often followed by intermittent pulse, especially during sleep, when the circulation is sluggish.

The cause of the rhythmic movements of the heart is debatable ground. That it is within the heart itself can scarcely

be questioned, for, when the heart of some animals is dissevered from all connections, and taken from the body, it may go on performing its rhythmical movements. Still, the quality and quantity of blood influence them in an unmistakable manner. The fact that shutting off supply of blood to the structure of the heart will arrest its contractions, was shown in 1842 by Mr. Erichsen. Dr. Brown-Séquard has attempted to explain the motion to be due to the carbonic acid present in the venous blood, and Dr. Radcliffe has also given a similar explanation.

The experiments of Dr. Paget show that the power causing rhythmical motion does not reside in all parts of the heart alike; that, in fact—

"If, for example, the cut-out heart (of any of the amphibia) be divided into two pieces, one comprising the auricles and the base of the ventricle, the other comprising the rest of the ventricle, the former will continue to act rhythmically, the latter will cease to do so, and no rhythmic action can be by any means excited in it. The piece of ventricle does not lose its power of motion, for if it be in any way stimulated, it contracts vigorously, but it never contracts without such an external stimulus, and when stimulated it never contracts more than once for each stimulus.

"Other sections of the heart, and experiments of other kinds, would show that the cause of the rhythmic action of the ventricle, and probably also of the auricles, so long as they are associated with it, and not with the venous trunks, is something in and near the boundary ring between the auricles and ventricles; for what remains connected with this ring, or grew with a part of it, in a longitudinally bisected heart, retains its rhythm, and what is disconnected from it loses its rhythm."

If we take a merely material view of the subject, no doubt we have arrived at the solution as nearly as we ever will. But is it useless or absurd to look further? The experiments of the great Harvey with the egg of the hen show that active life remains inchoate in the punctum saliens or

germinal spot until warmed into active life. This principle came into the egg organization at the time of its fecundation. Its first life-motion is rhythmical movement of particles before any portion of the heart's structure can be seen. The little red point appears and disappears rhythmically, and thus the *principle* builds its house, the auricle being its first chamber. The very nature of this principle is rhythmical. Its special home is in the ganglionic nervous system, but it pervades the whole body; wherever there is nerve-fibre accompanying the smallest capillary—the vaso-motor—it is present. Aberration from its normal life-action is disease; and influences, both outside and inside of the body, make impressions upon this life, helping to determine the character of the disease. Medicines act upon it, but their *modus operandi* is a sealed mystery. That they are purgative, emetic, stimulant, sedative, or alterative, we only know the fact. The heart, supplied with about three hundred ganglia, is the centre and citadel of this life, and its abnormal or disturbed action is sometimes mysterious evidence of both intrinsic and extrinsic disease.

Acoustic properties of the chest have not been dwelt upon as their importance demands. The diagnosis of murmurs within the chest is facilitated, or otherwise, according to its conditions as an acoustic chamber. The difficulty of hearing signs in the chest of a hunchback is recognized; it is also a well-known fact that, as the heart enlarges, the murmurs grow weaker, so that those which had been once easily detected become feeble, or disappear altogether. Still, they have been accounted for, it seems to me, upon every other principle than the true one.

In Dr. Cammann's last illness, by his request, I was called to examine him. After he had explained to me that I would find obstructive and regurgitant murmurs, of which he had been long cognizant, and of which he explained the cause and origin, and of their gradual increase, I found that I could but just hear the soft, feeble murmurs of aortic obstruction and regurgitation, but intra-ventricular murmurs were not heard. I told the doctor that the regurgitant murmur which he had

emphasized in relating the case, was slight: "Yes," he said, "it is but a chink." Dr. Peugnet told me that when he examined him at the beginning of his illness, the murmurs were loud and easily heard. I felt mortified that my ear had failed me, as I supposed, caused by a long ride in the cold, in an open carriage. The doctor had circumscribed pleuritis with effusion and pneumonia. In time the effusion was absorbed, and then the murmurs at the apex-beat were easily heard.

Another case, of which I have no notes, in which I failed to make out a murmur where it should have been heard, and which afterward returned, as the inter-current pneumonia, became convalescent, also annoyed me, and again I blamed my ear. Not long afterward I saw in the London *Medical Times and Gazette*, or in the London *Lancet*, the question, "Why do cardiac murmurs disappear during pneumonia or pleurisy?" I felt at once that the cause of my not hearing the murmurs more plainly in Dr. Cammann's case, as well as in that of this other patient, was because they were obscured by some cause I then did not know.

Other cases of cardiac murmurs disappearing or becoming obscured during the presence of pneumonia or pleuritis, led me to believe that it was in accordance with physical law. A patient with pleuritic effusion was sent to me by Dr. Otis for examination. I knew from a previous auscultation that he had aortic obstructive and aortic regurgitant murmurs. At this time, however, they could not be heard. I wrote to Dr. Otis, stating these facts, and predicting that when the effusion was absorbed these murmurs would again return, which proved to be the case.

On August 27, 1864, I saw Miss Hall, matron of the Home for Soldiers' Children, in Fifty-seventh Street near Eighth Avenue, with Drs. Charles McMillan, J. L. Smith, and E. Krakowizer. There were no heart-murmurs, but as all the rational signs of cardiac disease, with increased area of dullness under percussion, signified hypertrophy, it was suggested that we should examine her for pneumonia, and, upon raising

her up and listening behind, it was clearly made out. I then predicted that, when the pneumonia was well, we would be able to diagnosticate her cardiac disease. This was afterward done, and Dr. J. L. Smith took notes of the examination, and upon her death, some months afterward, was able to verify the diagnosis. He presented the heart, with history, to the Pathological Societ y, and a committee was appointe to examine into the facts concerning the disappearance of heart-murmurs during the presence of pneumonia and pleuritis, and to report. If my memory serves me, the committee reported in substance, in the summer of 1865, that in some cases observed in Bellevue Hospital, murmurs grew feeble or disappeared on the advent of pneumonia or pleurisy, and that it was their opinion this phenomenon was owing to the feebleness of the heart and its frequency, for in the cases noticed the pulse was 120 or more in a minute.

These reasons I had myself considered and rejected, for at the same time that Miss Hall was ill, I had another patient, O. B. H——, who had had for years a double murmur, which, when attacked with pneumonia, disappeared. His pulse ordinarily was about 50 in a minute, but during the pneumonia it rose as high as 80, but no higher. Drs. Chas. McMillan and J. L. Smith were also both cognizant of the facts as narrated. The philosophical explanation of these phenomena occurred to me during the winter of 1864–'65, with the following proof and illustration: The chest is a musical chamber, and may be represented by a violin. When the instrument is tuned and in order, its acoustic qualities may be considered as perfect. If a watch or music-box be placed within the violin, hanging from its roof, auscultation will reveal the slightest jar or noise made by the works of the watch, or bring out with distinctness the low tones of the music-box. But, if, while the ear or stethoscope is still placed upon the violin, water or sand be poured into its chamber, the sounds of the box or watch will grow feeble or disappear. The low notes of the music-box disappear entirely, as also does any jarring of the wheels of the watch. These phenomena are invariable

because they are the result of acoustic law. The application of physical law to art is to render it scientific, and scientific medicine is the immediate professional want of our time. If acoustic law is applied to auscultation in physical diagnosis, it will remove it from the domain of doubt or uncertainty, just so far as its principles are intelligently applied.